Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=AND		
L13	L12 and (PDGF)	2	L13
L12	L11 and (WAP or (beta adj lactoglobulin) or casein)	59	L12
L11	((transgenic adj non-human) adj mammal) same (milk)	116	L11
L10	L9 and (PDGF)	16	L10
L9	((transgenic adj non-human) adj mammal) and (milk or (mammary adj gland))	226	L9
L8	(PDGF or PDGF-AA or PDGF-BB or PDGF-AB) and (transgenic adj mammal)	42	L8
L7	(PDGF or PDGF-AA or PDGF-BB or PDGF-AB) same (transgenic adj mammal)	1	L7
L6	L5 and (mammary adj gland)	8	L6
L5	(PDGF or PDGF-AA or PDGF-BB or PDGF-BB) and (transgenic adj mammal)	42	L5
L4	(transgenically adj produced) adj (platelet adj derived)	0	L4
L3	(transgenically adj produced) adj (PDGF)	0	L3
L2	Meade-harry-M$.in.	9	L2
L1	Echelard-Yann.in.	3	L1

END OF SEARCH HISTORY

... family, the colony-stimulating factor (CSF) family, and a few other seemingly unrelated regulatory peptides, such as hepatocyte growth factor (HGF), platelet-derived growth factor (*PDGF*), and various interleukins, interferons and tumour necrosis factor-related proteins. In addition to the well-known effects on cell proliferation, these regulatory peptide factors regulate several other functional properties of epithelial and other cell populations, such as differentiation, migration, and extracellular matrix deposition and degradation. This *review* is designed not to discuss all the identified factors in detail but to highlight some of the basic principles of growth factor action in the...
?ds

Set	Items	Description
S1	146	(PDGF OR PDGF-AA OR PDGF-BB OR PDGF-AB) (S) (TRANSGENIC)
S2	0	S1 AND (WAP OR (BETA (W) LACTOGLOBULIN) OR CASEIN)
S3	0	S1 AND ((MAMMARY (W) GLAND) OR (MILK))
S4	0	S1 AND (TRANSGENIC (W) NON-HUMAN (W) MAMMAL)
S5	122	S1 AND (TRANSGENIC (W) (MOUSE OR MICE OR GOAT))
S6	68	RD (unique items)
S7	0	S6 AND (MILK OR BIOREACTOR)
S8	504	(PDGF OR PDGF-AA OR PDGF-BB OR PDGF-AB) AND (REVIEW)
S9	59	S8 NOT PY<2001
S10	50	RD (unique items)

?logoff

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       02jan03 09:56:23 User259876 Session D450.2
            $4.34    1.358 DialUnits File155
               $4.20  20 Type(s) in Format  3
            $4.20  20 Types
     $8.54  Estimated cost File155
            $6.75    1.205 DialUnits File5
     $6.75  Estimated cost File5
           $11.73    1.303 DialUnits File73
    $11.73  Estimated cost File73
            OneSearch, 3 files,  3.866 DialUnits FileOS
     $3.46  TELNET
    $30.48  Estimated cost this search
    $30.89  Estimated total session cost   3.971 DialUnits


### Status: Signed Off. (16 minutes)
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### Status: Path 1 of [Dialog Information Services via Modem]

### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS?
### Status: Signing onto Dialog
 ********
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
### Status: Connected


Dialog level 02.12.20D

Last logoff:  31dec02 10:12:08
Logon file001 02jan03 09:40:48
           *** ANNOUNCEMENT ***
                    ***
--File 515 D&B Dun's Electronic Business Directory is now online
completely updated and redesigned. For details, see HELP NEWS 515.
                    ***
--File 990 - NewsRoom now contains May 2002 to present records.
File 993 - NewsRoom archive contains 2002 records from January 2002-
April 2002. To search all 2002 records, BEGIN 990,993 or B NEWS2002.
                    ***
--Alerts have been enhanced to allow a single Alert profile to be
stored and run against multiple files. Duplicate removal is available
across files and for up to 12 months. The Alert may be run according
to the file's update frequency or according to a custom
calendar-based schedule. There are no additional prices for these
enhanced features. See HELP ALERT for more information.
                    ***
--U.S. Patents Fulltext (File 654) has been redesigned with
new search and display features.  See HELP NEWS 654 for
information.
                    ***
--Connect Time joins DialUnits as pricing options on Dialog.
See HELP CONNECT for information.
                    ***
--CLAIMS/US Patents (Files 340,341, 942) have been enhanced
with both application and grant publication level in a
single record. See HELP NEWS 340 for information.
                    ***
--SourceOne patents are now delivered to your email inbox
as PDF replacing TIFF delivery. See HELP SOURCE1 for more
information.
                    ***
--Important news for public and academic
libraries.  See HELP LIBRARY for more information.
                    ***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
                    ***
For information about the access to file 43 please see Help News43.
***
NEW FILES RELEASED
***Dialog NewsRoom - Current 3-4 months (File 990)
***Dialog NewsRoom - 2002 Archive (File 993)
***Dialog NewsRoom - 2001 Archive (File 994)
***Dialog NewsRoom - 2000 Archive (File 995)
***TRADEMARKSCAN-Finland (File 679)
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***TRADEMARKSCAN-Norway (File 678)
***TRADEMARKSCAN-Sweden (File 675)
***

UPDATING RESUMED
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***

RELOADED
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***Kompass Western Europe (File 590)
***D&B - Dun's Market Identifiers (File 516)

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***ISMEC: Mechanical Engineering Abstracts (File 14)
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***Water Resources Abstracts (File 117)
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***Fort Lauderdale Sun Sentinel (File 497)
***The Orlando Sentinel (File 705)
***Newport News Daily Press (File 747)
***U.S. Patents Fulltext 1980-1989 (File 653)
***Washington Post (File 146)
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***Court Filings (File 793)
***Publishers, Distributors & Wholesalers of the U.S. (File 450)
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***Tax Notes Today (File 790)
***Worldwide Tax Daily (File 792)

New document supplier
IMED has been changed to INFOTRIE (see HELP OINFOTRI)

>>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
>>> of new databases, price changes, etc. <<<
****
KWIC is set to 50.
HILIGHT set on as '*'
* ** **

File 1:ERIC 1966-2002/Dec 13
(c) format only 2002 The Dialog Corporation

Set	Items	Description

Cost is in DialUnits
?b 155, 5, 73

```
      02jan03 09:41:00 User259876 Session D450.1
           $0.37    0.105 DialUnits File1
     $0.37  Estimated cost File1
     $0.04  TELNET
     $0.41  Estimated cost this search
     $0.41  Estimated total session cost   0.105 DialUnits


SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R)  1966-2002/Nov W4
*File 155: Updating of completed records has resumed.See Help News155.
Alert feature enhanced with customized scheduling. See HELP ALERT.
  File   5:Biosis Previews(R)  1969-2002/Dec W5
         (c) 2002 BIOSIS
*File   5: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.
  File  73:EMBASE  1974-2002/Dec W4
         (c) 2002 Elsevier Science B.V.
*File  73: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.

      Set  Items  Description
      ---  -----  -----------
?s (PDGF or PDGF-AA or PDGF-BB or PDGF-AB) (s) (transgenic)
           20771  PDGF
               7  PDGF-AA
              29  PDGF-BB
               8  PDGF-AB
          110112  TRANSGENIC
      S1     146  (PDGF OR PDGF-AA OR PDGF-BB OR PDGF-AB) (S) (TRANSGENIC)
?s s1 and (WAP or (beta (w) lactoglobulin) or casein)
             146  S1
             731  WAP
         1410330  BETA
            6678  LACTOGLOBULIN
            6422  BETA(W)LACTOGLOBULIN
           49646  CASEIN
      S2       0  S1 AND (WAP OR (BETA (W) LACTOGLOBULIN) OR CASEIN)
?s s1 and ((mammary (w) gland) or (milk))
             146  S1
          121212  MAMMARY
          408092  GLAND
           28924  MAMMARY(W)GLAND
          177809  MILK
      S3       0  S1 AND ((MAMMARY (W) GLAND) OR (MILK))
?s s1 and (transgenic (w) non-human (w) mammal)
             146  S1
          110112  TRANSGENIC
              70  NON-HUMAN
          128536  MAMMAL
               0  TRANSGENIC(W)NON-HUMAN(W)MAMMAL
      S4       0  S1 AND (TRANSGENIC (W) NON-HUMAN (W) MAMMAL)
?s s1 and (transgenic (w) (mouse or mice or goat))
             146  S1
          110112  TRANSGENIC
         1378281  MOUSE
         1241601  MICE
           44636  GOAT
           57017  TRANSGENIC(W)((MOUSE OR MICE) OR GOAT)
      S5     122  S1 AND (TRANSGENIC (W) (MOUSE OR MICE OR GOAT))
?rd
...examined 50 records   (50)
...examined 50 records   (100)
...completed examining records
      S6      68  RD (unique items)
?
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PLEASE ENTER A COMMAND OR [redacted]E LOGGED OFF IN 5 MINUTES
?s s6 and (milk or bioreactor)
68 S6
177809 MILK
17007 BIOREACTOR
S7 0 S6 AND (MILK OR BIOREACTOR)
?t s6/3,k/1-10

6/3,K/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13739609 22206300 PMID: 12217216
Lung pathology in platelet-derived growth factor *transgenic* *mice*: effects of genetic background and fibrogenic agents.
Li Jian; Ortiz Luis A; Hoyle Gary W
Section of Pulmonary Diseases, Critical Care and Environmental Medicine, Department of Medicine and Interdisciplinary Graduate Program in Molecular and Cellular Biology, Tulane University Health Sciences Center, New Orleans, Louisiana, USA.
Experimental lung research (United States) Sep 2002, 28 (6) p507-22, ISSN 0190-2148 Journal Code: 8004944
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Lung pathology in platelet-derived growth factor *transgenic* *mice*: effects of genetic background and fibrogenic agents.
We previously developed *transgenic* *mice* expressing human platelet-derived growth factor B chain (*PDGF*-B) from the lung-specific surfactant protein C (SPC) promoter. These mice developed enlarged airspaces, inflammation, and fibrosis of varying severity. In the present study we examined potential causes of this phenotypic variation and tested whether constitutive *PDGF* -B expression exacerbated fibrosis induced by bleomycin and silica. The SPC-PDGFB transgene construct was modified by replacement of the *PDGF*-B 3' UTR, which contains motifs known to mediate instability in other cytokine genes, with SV40 sequences containing an intron and polyadenylation signal. This modification...

...strains resulted in a more severe phenotype in SJL-bred mice compared to C57BL/6-bred mice after 4 generations. To determine whether SPC-PDGFB *transgenic* *mice* had increased susceptibility to fibrogenic agents, the mice were treated with bleomycin or silica. No significant differences were detected in lung weight, hydroxyproline content, or histopathologic changes between *transgenic* and wild-type mice after bleomycin or silica treatment. These results demonstrate that the amount of *PDGF*-BB produced in wild-type mice is not a limiting factor in the development of bleomycin- or silica-induced pulmonary fibrosis.

6/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13489710 22130990 PMID: 12135350
Generation and characterization of two *transgenic* *mouse* lines expressing human ApoE2 in neurons and glial cells.
Georgopoulos Spiros; McKee Ann; Kan Horng-Yuan; Zannis Vassilis I
Section of Molecular Genetics, Whitaker Cardiovascular Institute, Department of Medicine, Boston University School of Medicine, Boston, Massachusetts 02118, USA.
Biochemistry (United States) Jul 30 2002, 41 (30) p9293-301, ISSN 0006-2960 Journal Code: 0370623
Contract/Grant No.: AG-12717; AG; NIA; HL68216; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed

Generation and characterization of two *transgenic* *mouse* lines expressing human ApoE2 in neurons and glial cells.

...disease (AD). The epsilon 4 and epsilon 2 alleles have been associated with increased and decreased risk for AD, respectively. We have generated and characterized *transgenic* *mice* in which the human apoE2 gene is expressed under the control of the platelet-derived growth factor B-chain (*PDGF* -B) promoter, or the transferrin (TF) promoter. S1 nuclease analysis and immunoblotting showed that the *PDGF* -B apoE2 mice express apoE2 exclusively in the brain whereas the TF apoE2 mice express apoE2 in the liver and in the brain. In the TF apoE2 mouse line, apoE2 is also detected in the plasma. The *PDGF*-B apoE2 and the TF apoE2 *transgenic* *mice* were bred back to apoE(-)(/)(-) background. Immunohistochemical analysis showed that the *PDGF* apoE2 x apoE(-)(/)(-) and the TF apoE2 x apoE(-)(/)(-) mice express human apoE2 within the neocortex in hippocampal neurons and glial cells, respectively. ApoE(-)(/)(-) mice...

... age-dependent loss of synaptophysin. Immunoblotting of mouse brain extracts and immunohistochemical analysis of brain sections showed that apoE expression in both apoE2 x apoE(-)(/)(-) *transgenic* lines was associated with significant recovery of brain synaptophysin levels as compared to the levels of apoE(-)(/)(-) littermates of the same age. These apoE2-expressing mice, when bred back on amyloid precursor protein (APP) *transgenic* *mice* or other mouse lines featuring alterations in lipoprotein metabolism, may provide new mouse models for elucidating the role of apoE2 in lipid homeostasis in the...

6/3,K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13454582 22106606 PMID: 12111846

Differential neuropathological alterations in *transgenic* *mice* expressing alpha-synuclein from the platelet-derived growth factor and Thy-1 promoters.

Rockenstein Edward; Mallory Margaret; Hashimoto Makoto; Song David; Shults Clifford W; Lang Ingrid; Masliah Eliezer

Department of Neurosciences, University of California San Diego, La Jolla, California 92093-0624, USA.

Journal of neuroscience research (United States) Jun 1 2002, 68 (5) p568-78, ISSN 0360-4012 Journal Code: 7600111

Contract/Grant No.: AG10869; AG; NIA; AG18440; AG; NIA; AG5131; AG; NIA

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Differential neuropathological alterations in *transgenic* *mice* expressing alpha-synuclein from the platelet-derived growth factor and Thy-1 promoters.

...associated with neurodegenerative disorders, such as Lewy body disease and multiple system atrophy. We previously showed that expression of wild-type human alpha-synuclein in *transgenic* *mice* results in motor and dopaminergic deficits associated with inclusion formation. To determine whether different levels of human alpha-synuclein expression from distinct promoters might result in neuropathology mimicking other synucleopathies, we compared patterns of human alpha-synuclein accumulation in the brains of *transgenic* *mice* expressing this molecule from the murine Thy-1 and platelet-derived growth factor (*PDGF*) promoters. In murine Thy-1-human alpha-synuclein *transgenic* *mice*, this protein accumulated in synapses and neurons throughout the brain, including the thalamus, basal ganglia, substantia nigra, and brainstem. Expression of human alpha-synuclein from the *PDGF* promoter resulted in accumulation in synapses of the neocortex, limbic system, and olfactory regions as well as formation of inclusion bodies in neurons in deeper...

... alpha-synuclein expression in glial cells mimicking some features of multiple system atrophy. These results show a more widespread accumulation of human alpha-synuclein in *transgenic* *mouse* brains. Taken together, these studies support the contention that human alpha-synuclein expression in *transgenic* *mice* might mimic some neuropathological alterations observed in Lewy body disease and other synucleopathies, such as multiple system atrophy. Copyright 2002 Wiley-Liss, Inc.

6/3,K/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13288397 22033198 PMID: 12037011
Retina-specific expression of PDGF-B versus PDGF-A: vascular versus nonvascular proliferative retinopathy.
Mori Keisuke; Gehlbach Peter; Ando Akira; Dyer Gawain; Lipinsky Evan; Chaudhry Aneeka G; Hackett Sean F; Campochiaro Peter A
Department of Ophthalmology, The Johns Hopkins University School of Medicine, Baltimore, Maryland 21287-9277, USA.
Investigative ophthalmology & visual science (United States) Jun 2002, 43 (6) p2001-6, ISSN 0146-0404 Journal Code: 7703701
Contract/Grant No.: EY-05951; EY; NEI; EY-12609; EY; NEI; K08 EY-13420; EY; NEI; P30-EY-1765; EY; NEI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

PURPOSE: Platelet-derived growth factor (*PDGF*) has been implicated in vascular proliferative retinopathies, such as diabetic retinopathy, and in nonvascular retinopathies, such as proliferative vitreoretinopathy. Traction retinal detachment is a central feature of both types of disease. Hemizygous rhodopsin promoter/*PDGF*-B (rho/*PDGF*-B) *transgenic* *mice* exhibit proliferation of vascular cells, glia, and retinal pigmented epithelial (RPE) cells, resulting in traction retinal detachment. Hemizygous rho/*PDGF*-A *transgenic* *mice* show mild proliferation of glial cells and no traction retinal detachments. This study was undertaken to determine whether higher levels of endogenously produced *PDGF*-A in the retinas of mice result in retinal detachment. METHODS: To achieve high-level expression of *PDGF*-A in the retina, homozygous rho/*PDGF*-A (rho/*PDGF* -AA) mice were generated. The phenotype of these mice was compared with that of homozygous rho/*PDGF*-B (rho/*PDGF*-BB) mice and double hemizygous rho/*PDGF*-B-rho/*PDGF*-A (rho/*PDGF*-AB) mice. RESULTS: Rho/*PDGF*-BB and rho/*PDGF* -AB mice showed a phenotype similar to that previously described in rho/*PDGF* -B mice. There was extensive proliferation of glial and vascular cells, resulting in fibrovascular membranes that detached the retina. *PDGF* -AA mice showed extensive proliferation of glial cells and traction retinal detachment. CONCLUSIONS: High retinal expression of *PDGF*-A results in extensive proliferation of glial cells and traction retinal detachment without vascular cell involvement, similar to proliferative vitreoretinopathy in humans. High retinal expression of *PDGF*-B results in traction retinal detachment from proliferation of both vascular and nonvascular cells, similar to diabetic retinopathy in humans.

6/3,K/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13277050 21886334 PMID: 11888678
Experimental models of growth factor-mediated angiogenesis and blood-retinal barrier breakdown.
Vinores S A; Seo M S; Okamoto N; Ash J D; Wawrousek E F; Xiao W H; Hudish T; Derevjanik N L; Campochiaro P A
Wilmer Eye Institute, Johns Hopkins University School of Medicine, 825

Maumenee Building, 600 North Wolfe Street, 21287-92 Baltimore, MD 21287-9289, USA. svinores@jhmi.edu
General pharmacology (England) Nov 2000, 35 (5) p233-9, ISSN 0306-3623 Journal Code: 7602417
Contract/Grant No.: EY05951; EY; NEI; EY10017; EY; NEI; EY12190; EY; NEI
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... results in neovascularization (NV) that originates from the vascular bed closest to the ganglion cell layer. To study the effects of VEGF, independent lines of *transgenic* *mice* that express VEGF in the lens and in the retina have been generated. Expression in the lens results in excessive proliferation and accumulation of angioblasts...
... blood vessel organization or maturation in the prenatal mouse. Abnormal vessels do form on the retinal surface, but not until the second postnatal week. In *transgenic* *mice* expressing VEGF in the photoreceptors, NV originates from the deep capillary bed--the vascular bed closest to the photoreceptors. NV is accompanied by localized blood-retinal barrier breakdown. NV is also induced in *PDGF*-B *transgenic* *mice*. *PDGF*-B expression in the lens occurs prenatally and, during this time, mainly affects the perilenticular vessels. Postnatally, *transgenic* *mice* expressing *PDGF* -B in the lens or photoreceptors show a similar phenotype. In both models, a highly vascularized cell mass containing endothelial cells, pericytes, and glia forms...

6/3,K/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13219469 21977610 PMID: 11980881
Intraocular adenoviral vector-mediated gene transfer in proliferative retinopathies.
Mori Keisuke; Gehlbach Peter; Ando Akira; Wahlin Karl; Gunther Vicky; McVey Duncan; Wei Lisa; Campochiaro Peter A
Departments of Ophthalmology and Neuroscience, The Johns Hopkins University School of Medicine, Maumenee 719, 600 N. Wolfe Street, Baltimore, MD 21287-9277, USA.
Investigative ophthalmology & visual science (United States) May 2002, 43 (5) p1610-5, ISSN 0146-0404 Journal Code: 7703701
Contract/Grant No.: EY 05951; EY; NEI; EY 12609; EY; NEI; K08 EY 13420; EY; NEI; P30 EY 1765; EY; NEI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... RPE cells. Expression of LacZ after intravitreous injection of AdLacZ.10 was significantly greater in mice with two types of proliferative retinopathy (ischemic retinopathy or *transgenic* *mice* with retina-specific expression of platelet-derived growth factor (*PDGF*)-BB or *PDGF* -AB) than littermate control animals. Cells within epiretinal membranes and activated Muller cells were preferentially transduced in eyes with proliferative retinopathy. CONCLUSIONS: These data suggest...

6/3,K/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12789025 21643886 PMID: 11784039
Overexpression of *PDGF*-A in the lung epithelium of *transgenic* *mice* produces a lethal phenotype associated with hyperplasia of mesenchymal cells.
Li J; Hoyle G W
Section of Pulmonary Diseases, Critical Care and Environmental Medicine,

Department of Medicine, Tulane University Health Sc ces Center, New Orleans, Louisiana 70112, USA.
Developmental biology (United States) Nov 15 2001, 239 (2) p338-49, ISSN 0012-1606 Journal Code: 0372762
Contract/Grant No.: HL58610; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Overexpression of *PDGF*-A in the lung epithelium of *transgenic* *mice* produces a lethal phenotype associated with hyperplasia of mesenchymal cells.

Transgenic *mice* expressing platelet-derived growth factor A chain (*PDGF*-A) in the distal lung epithelium from the surfactant protein C (SPC) promoter were generated to investigate the role of this growth factor in lung...

... Histologic analysis of embryonic day (E) 16.5 lungs revealed increased mesenchymal cells and acinar buds and decreased bronchioles and dilated airspaces in SPC-PDGFA *transgenic* *mice*. At E18.5, nontransgenic lungs exhibited lung morphology typical of the saccular stage of lung development, including dilated airspaces, thin respiratory epithelium and mesenchyme, and elastin fiber deposition in primary septa. In contrast, E18.5 *transgenic* lungs retained many features of the canalicular stage of lung development, including undilated airspaces, cuboidal respiratory epithelium, thickened mesenchyme, and lack of parenchymal elastin deposition. These results indicate that *PDGF*-A is a potent growth factor for mesenchymal cells in the developing lung and that the downregulation of *PDGF* -A expression that normally occurs in the lung during late gestation is required for transition from the canalicular to the saccular stage of lung development...

6/3,K/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11289613 21324430 PMID: 11431731

Roles of Akt/PKB and IKK complex in constitutive induction of NF-kappaB in hepatocellular carcinomas of transforming growth factor alpha/c-myc *transgenic* *mice*.

Factor V; Oliver A L; Panta G R; Thorgeirsson S S; Sonenshein G E; Arsura M
Laboratory of Experimental Carcinogenesis, Division of Basic Sciences, National Cancer Institute, Bethesda, MD, USA.
Hepatology (Baltimore, Md.) (United States) Jul 2001, 34 (1) p32-41, ISSN 0270-9139 Journal Code: 8302946
Contract/Grant No.: CA36355; CA; NCI; CA78616; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Roles of Akt/PKB and IKK complex in constitutive induction of NF-kappaB in hepatocellular carcinomas of transforming growth factor alpha/c-myc *transgenic* *mice*.

... HCCs) derived from bitransgenic mice harboring TGF-alpha and c-myc transgenes targeted specifically to the liver were compared with HCCs from c-myc single *transgenic* *mice* . Tumors from bitransgenic mice are characterized by a higher frequency of appearance, lower apoptotic index, and a higher rate of cell proliferation. Here we show that NF-kappaB is activated in HCCs of double TGF-alpha/c-myc *transgenic* *mice*, but not of c-myc single *transgenic* *mice* , suggesting that TGF-alpha mediates induction of NF-kappaB. Activation of the IKK complex was observed in the HCCs of double TGF-alpha/c-myc *transgenic* *mice* , implicating this pathway in NF-kappaB induction. Lastly, activation of the Akt/protein

kinase B (PKB), which has recently been implicated in NF-kappaB activation by *PDGF* , TNF-alpha, and Ras, was also observed. Importantly, human HCC cell lines similarly displayed NF-kappaB activation. Thus, these studies elucidate an anti-apoptotic mechanism...

6/3,K/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11133530 21148081 PMID: 11250151
Control of progenitor cell number by mitogen supply and demand.
van Heyningen P; Calver A R; Richardson W D
Wolfson Institute for Biomedical Research and Department of Biology, University College London, Gower Street, WC1E 6AE, London, UK.
Current biology : CB (England) Feb 20 2001, 11 (4) p232-41, ISSN 0960-9822 Journal Code: 9107782
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... by cumulative BrdU labeling in vivo that the progenitor cell division cycle slows down markedly as their numbers increase during embryogenesis. When cultured in saturating *PDGF*, the main mitogen for these cells, their cell cycle accelerated and was independent of their prior rate of division in vivo. This shows that mitogens are limiting in vivo, and suggests that division normally slows down because the *PDGF* concentration declines. In *PDGF*-*transgenic* *mice*, cell number was proportional to the *PDGF* supply and apparently unsaturable; at ten times the normal rate of supply, cell number was still increasing but the animals were no longer viable. CONCLUSIONS: Progenitor cell proliferation in the embryo is limited by environmental factors, not a cell-intrinsic mechanism. The linear relationship between *PDGF* supply and final cell number strongly suggests that cells deplete the mitogenic activity in their environment at a rate proportional to the total number of cells. The cells might simply consume the available *PDGF* or they might secrete autocrine inhibitors, or both.

6/3,K/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11121706 21128120 PMID: 11222634
Alpha-1-antichymotrypsin promotes beta-sheet amyloid plaque deposition in a *transgenic* *mouse* model of Alzheimer's disease.
Nilsson L N; Bales K R; DiCarlo G; Gordon M N; Morgan D; Paul S M; Potter H
Suncoast Gerontology Center, Department of Biochemistry and Molecular Biology, Moffitt Cancer Center, College of Medicine, University of South Florida, Tampa, Florida 33612, USA. lnilsson@hsc.usf.edu
Journal of neuroscience : the official journal of the Society for Neuroscience (United States) Mar 1 2001, 21 (5) p1444-51, ISSN 1529-2401 Journal Code: 8102140
Contract/Grant No.: AG09665; AG; NIA
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Alpha-1-antichymotrypsin promotes beta-sheet amyloid plaque deposition in a *transgenic* *mouse* model of Alzheimer's disease.
... has been shown to catalyze amyloid beta-peptide polymerization in vitro. We have investigated the impact of ACT on amyloid deposition in vivo by generating *transgenic* GFAP-ACT-expressing mice and crossing them with the *PDGF* -hAPP/V717F mice, which deposit amyloid in an age-dependent manner. The number of amyloid deposits measured by Congo Red birefringence was increased in the double ACT/amyloid precursor protein (APP)

transgenic *mice* co red with *transgenic* *mice* t only expressed APP, particularly in the hippocampus where ACT expression was highest, and the increase was preceded by elevated total amyloid beta-peptide levels...
?ds

Set	Items	Description
S1	146	(PDGF OR PDGF-AA OR PDGF-BB OR PDGF-AB) (S) (TRANSGENIC)
S2	0	S1 AND (WAP OR (BETA (W) LACTOGLOBULIN) OR CASEIN)
S3	0	S1 AND ((MAMMARY (W) GLAND) OR (MILK))
S4	0	S1 AND (TRANSGENIC (W) NON-HUMAN (W) MAMMAL)
S5	122	S1 AND (TRANSGENIC (W) (MOUSE OR MICE OR GOAT))
S6	68	RD (unique items)
S7	0	S6 AND (MILK OR BIOREACTOR)

```
?s (PDGF or PDGF-AA or PDGF-BB or PDGF-AB) and (review)
          20771  PDGF
              7  PDGF-AA
             29  PDGF-BB
              8  PDGF-AB
        1296993  REVIEW
      S8     504  (PDGF OR PDGF-AA OR PDGF-BB OR PDGF-AB) AND (REVIEW)
?s s8 not py<2001
Processing
Processing
            504  S8
       32358064  PY<2001
      S9     59  S8 NOT PY<2001
?rd
...examined 50 records  (50)
...completed examining records
     S10     50  RD (unique items)
?t s10/3,k/1-10
```

10/3,K/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

14029536 22311687 PMID: 12424751

Mesangial cell proliferation inhibitors for the treatment of proliferative glomerular disease.

Kurogi Yasuhisa; et al

Otsuka Pharmaceutical Co., Ltd., R&D Alliances, 463-10, Kagasuno, Kawauchi-cho, Tokushima 771-0192, Japan.

Medicinal research reviews (United States) Jan 2003, 23 (1) p15-31, ISSN 0198-6325 Journal Code: 8103150

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: In Process

... mesangium and glomerulosclerosis. Reduction of MC proliferation in glomerular disease models by treatment with heparin, low-protein diet, or antibodies to platelet-derived growth factor (*PDGF*), have been shown to reduce extracellular matrix expansion and glomerulosclerotic changes. Therefore, MC proliferation inhibitors may offer therapeutic opportunities for the treatment of proliferative glomerular...

... that the MC proliferation is inhibited by many kinds of pharmacological drugs, for example, angiotensin converting enzyme (ACE) inhibitors, leukotriene D(4) (LTD(4)) antagonists, *PDGF* inhibitors, matrix metalloproteinases (MMP) inhibitors, 3-hydroxy-3 methyl glutaryl-coenzymeA (HMG-CoA) inhibitors, cyclin-dependent kinases (CDK) inhibitors, and others. This *review* summarizes the recently reported MC proliferation inhibitors with their pharmacological properties on the basis of their chemical structures. Copyright 2002 Wiley Periodicals, Inc. Med Res...

10/3,K/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

14024109 22305654 PMID: 12418561
Bone growth factors in maxillofacial skeletal reconstruction.
Schilephake H; et al
International journal of oral and maxillofacial surgery (Denmark) Oct 2002, 31 (5) p469-84, ISSN 0901-5027 Journal Code: 8605826
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: In Process

A literature *review* was performed to survey the available information on the potential of bone growth factors in skeletal reconstruction in the maxillofacial area. The aim of this *review* was to characterize the biological and developmental nature of the growth factors considered, their molecular level of activity and their osteogenic potential in craniofacial bone...

... selected for evaluation by the content of the abstracts. All growth factors considered have a fundamental role in growth and development. In postnatal skeletal regeneration, *PDGF* plays an important role in inducing proliferation of undifferentiated mesenchymal cells. It is an important mediator for bone healing and remodelling during trauma and infection...

... craniofacial skeletal defects has not yet shown a clear potential for enhancement of bone regeneration in the reported dosages. The combination of IGF-I with *PDGF* has been effective in promoting bone regeneration in dentoalveolar defects around implants or after periodontal bone loss. TGFbeta alone in skeletal reconstruction appears to be...

10/3,K/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13984638 22260886 PMID: 12373680
[Inflammatory mechanisms, arteriosclerosis and ischemic stroke: clinical data and perspectives]
Mecanismos inflamatorios, arteriosclerosis e ictus isquemico: datos de interes clinico y perspectivas.
Alvaro Gonzalez L C; Freijo Guerrero M M; Sadaba Garay F; et al
Basurtuko Ospitalea, Bilbao, Espa a.
Revista de neurologia (Spain) Sep 1 2002, 35 (5) p452-62, ISSN 0210-0010 Journal Code: 7706841
Document type: Journal Article
Languages: SPANISH
Main Citation Owner: NLM
Record type: In Process

... developed countries by causing ischemic cardiopathic and stroke. The ischemic atherotrombotic stroke is the most frequent form of the last one. In this sense we *review* herein the mechanisms underlying the artherosclerotic process. DEVELOPMENT. It is understood as an inflammatory disease, by taking into account the widely accepted hypothesis by Ross...

... IL 1, the TNF a and linfocitary ligands like the CD 40, or with antiinflammatory activity like the gamma interpheron; 3) Growth factors, with plaquetary (*PDGF*) and fibroblastic (FGF) variants as the cornerstone; 4) Markers of systemic inflammation, overall plasma C reactive protein and fibrinogen, that predict the risk of stroke...

10/3,K/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13953655 22211956 PMID: 12224049

LPA as a determinant of mesangial growth and apoptosis.
Inoue Chiyoko N; et al
Department of Pediatrics, Japanese Red Cross Sendai Hospital, Sendai, Japan. cnagano@sendai.jrc.or.jp
Seminars in nephrology (United States) Sep 2002, 22 (5) p415-22, ISSN 0270-9295 Journal Code: 8110298
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... feature of progression in many forms of renal diseases, including immunoglobulin A nephropathy, lupus nephritis, hemolytic uremic syndrome, and diabetic nephropathy. Platelet-derived growth factor (*PDGF*) has received much attention as the major mediator of mesangial cell proliferation by autocrine/paracrine mechanisms involving up-regulation of mesangial *PDGF* and its receptor on mesangial cells. In this *review*, we wish to spotlight lysophosphatidic acid (LPA), which in combination with *PDGF*, undoubtedly plays a key role as an autocrine and paracrine mediator in regulating mesangial cell growth. We not only showed that *PDGF* acts as a bimodal molecule for mesangial cells, inducing mesangial cell proliferation and death simultaneously, but also showed that LPA is a survival factor suppressing *PDGF*-induced mesangial cell death, thereby remarkably enhancing mesangial mitogenic response by *PDGF*. We believe that a better understanding of the mechanisms of mesangial cell proliferation by the combined action of *PDGF* and LPA could lead to novel diagnostic as well as therapeutic strategies, and thus help to better control proliferative glomerulonephritis. Copyright 2002, Elsevier Science (USA...

10/3,K/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13488954 22065004 PMID: 12069819
Sphingosine 1-phosphate signalling and termination at lipid phosphate receptors.
Pyne Susan; Pyne Nigel J
Department of Physiology and Pharmacology, Strathclyde Institute for Biomedical Sciences, University of Strathclyde, 27 Taylor Street, Scotland, Glasgow, UK. susan.pyne@strath.ac.uk
Biochimica et biophysica acta (Netherlands) May 23 2002, 1582 (1-3) p121-31, ISSN 0006-3002 Journal Code: 0217513
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... of how S1P can act as an intercellular mediator by binding to a new class of G-protein coupled receptors to regulate cell function. This *review* focuses on the enzymatic regulation of S1P formation and degradation and its interaction with a novel tethered receptor complex containing the S1P receptor (S1P(1)) and the platelet-derived growth factor (*PDGF*) beta receptor. This tethered receptor complex enables coincident integrative signalling to p42/p44 MAPK. This is compared with a sequential model in which *PDGF* promotes S1P release, which in turn acts on S1P(1) to promote Rac signalling.

10/3,K/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13380626 22083013 PMID: 12087679
Pathogenesis of Dupuytren's contracture--a *review*]
Patogeneza przykurczu Dupuytrena.
Kozma Ewa M; Olczyk Krystyna; Bobinski Rafal; Kasperczyk Mariusz; Szpyra

Katarzyna
Katedra i Zaklad Chemii Klinicznej i Diagnostyki Laboratoryjnej, Slaska Akademia Medyczna, Sosnowiec.
Chirurgia narzadow ruchu i ortopedia polska (Poland) 2002, 67 (1) p73-9, ISSN 0009-479X Journal Code: 2985137R
Document type: Journal Article; Review; Review, Tutorial ; English Abstract
Languages: POLISH
Main Citation Owner: NLM
Record type: Completed

Pathogenesis of Dupuytren's contracture--a *review*]
... and are a source of palmar contracture. The pivotal factors involved in changes of palmar fibroblasts functions seem to be growth factors (mainly TGF beta, *PDGF* and bFGF). However, the participation of reactive forms of oxygen in mentioned process is also considered.

10/3,K/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13211330 21976576 PMID: 11980191
Histological and immunohistochemical events during placentation in pigs.
Dantzer V; Winther H
Department of Anatomy and Physiology, Royal Veterinary and Agricultural University, Groennegaardsvej 7, DK-1870 Frederiksberg C, Denmark. Vibeke.Dantzer@iaf.kvl.dk
Reprod Suppl (England) 2001, 58 p209-22, Journal Code: 101142074
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... number of factors, the ligands and their receptors, such as insulin-like growth factor (IGF), transforming growth factor beta (TGF beta), platelet-derived growth factor (*PDGF*) and vascular endothelial growth factor (VEGF), as well as retinoids and calcium, is described. The comparison between these factors gives a strong impression of their complex interactions and hormonal relationships during placentation and vascular development in pigs. This *review* also emphasizes that retinoids are of great importance for placental function and that the transport of vitamin A appears to take place in the areolar...

10/3,K/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12996761 21625836 PMID: 11750857
***PDGF* and the testis.**
Mariani Stefania; Basciani Sabrina; Arizzi Mario; Spera Giovanni; Gnessi Lucio
Dept Medical Physiopathology, Policlinico Umberto I, University of Rome 'La Sapienza', 00161, Rome, Italy.
Trends in endocrinology and metabolism: TEM (United States) Jan-Feb 2002, 13 (1) p11-7, ISSN 1043-2760 Journal Code: 9001516
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

***PDGF* and the testis.**
... molecules and hormones that interact, often acting within short time windows, via reciprocal control relationships. The identification in the testis of platelet-derived growth factor (*PDGF*), a key regulator of connective tissue cells in embryogenesis and pathogenesis, has focused attention on the role of this growth factor in testicular pathophysiology.

This *review* summarize[redacted] recent advances in the study the actions of *PDGF* in the male gonad, and attempts to incorporate complex in vitro and in vivo experimental data into a model that might clarify the role played by *PDGF* in the mammalian testis.

10/3,K/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12850808 21479732 PMID: 11595457
Gene expression and gene therapy in experimental duodenal ulceration.
Szabo S; Deng X; Khomenko T; Yoshida M; Jadus M R; Sandor Z; Gombos Z; Matsumoto H
Path. & Lab. Med. Service, VA Medical Center, 5901 E. 7th Street, Long Beach, CA 90822-5201, USA. sandor.szabo@med.va.gov
Journal of physiology, Paris (France) Jan-Dec 2001, 95 (1-6) p325-35 , ISSN 0928-4257 Journal Code: 9309351
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... in gene expression may provide new mechanistic insights. Previously, we demonstrated that angiogenic growth factors are potent ulcer healing agents, and the synthesis of bFGF, *PDGF* and VEGF is enhanced early in duodenal ulcer healing. The initial molecular event in duodenal ulceration seems to be the organ-specific early release of ET-1 in the pre-ulcerogenic stages after the administration of duodenal ulcerogen cysteamine in rats. We also briefly *review* here data from literature indicating a central role of ET-1 in gastroduodenal ulceration. After studying the involvement of immediate early genes (e.g. egr...

... we now investigated expression of other genes in the duodenal mucosa in the early stages of chemically induced duodenal ulceration in rats. Following a brief *review* of principles of gene expression and gene therapy, we *review* our preliminary gene expression studies, involving monitoring about 1200 genes which revealed about 160 signals and prominent changes in about 30 genes in the early...

...while transcription factors (MAX, STAT 3) showed increased expression in 12 h. We recently also initiated gene therapy studies to enhance the local synthesis of *PDGF* and VEGF to accelerate duodenal ulcer healing, using a single dose of naked DNA (ND) or adenoviral (AV) vectors of VEGF and *PDGF* in rats with cysteamine-induced duodenal ulcers. Gene therapy with ND or AV of VEGF or *PDGF* significantly accelerated chronic duodenal ulcer healing, and increased levels of VEGF and *PDGF* were detected by Western blotting and ELISA in duodenal mucosa after both VEGF and *PDGF* gene therapy. Thus, gene expression studies provide new insights into the molecular mechanisms of duodenal ulceration and VEGF or *PDGF* gene therapy seems to be a new option to achieve a rapid ulcer healing.

10/3,K/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11320687 21367664 PMID: 11474304
Peptide growth factors in the intestine.
Dignass A U; Sturm A
Department of Medicine, Division of Hepatology and Gastroenterology, Charite-Campus Virchow Clinic, Berlin, Germany. axel.dignass@charite.de
European journal of gastroenterology & hepatology (England) Jul 2001, 13 (7) p763-70, ISSN 0954-691X Journal Code: 9000874
Document type: Journal Article; Review; Review, Academic
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed